PRINCIPLES AND PRACTICE OF

INFECTIOUS DISEASES

THIRD EDITION

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163. UREAPLASMA UREALYTICUM (T-STRAIN MYCOPLASMA) AND MYCOPLASMA HOMINIS

DAVID TAYLOR-ROBINSON

CHARACTERISTICS, ISOLATION, AND IDENTIFICATION

Of the seven mycoplasma species isolated from the human genital tract (Table 1), *Ureaplasma urealyticum* and *Mycoplasma hominis* are found most frequently. *Ureaplasma urealyticum* is one of three species in the genus *Ureaplasma*, and *M. hominis* is one of 77 species in the genus *Mycoplasma*, within the family Mycoplasmataceae and the class Mollicutes (see Chapter 162). The general properties noted in Table 1 of Chapter 161 for the mycoplasmas are also exhibited by *U. urealyticum* organisms (referred to as ureaplasmas) and *M. hominis*. Properties that distinguish these two microorganisms from each other, in addition to those they share with other mycoplasmas that are found less frequently in the genital tract, are presented in Table 1.

Detection of mycoplasmas in the genital tract depends on culturing specimens on appropriate media and identifying the isolates.[1] The medium most often used, as for other mycoplasmas, comprises a beefheart infusion broth, available commercially as pleuropneumonia-like organism (PPLO) broth, supplemented with fresh yeast extract (10 percent vol/vol; 25 percent wt/vol) and horse serum (20 percent vol/vol). However, the recovery of *M. hominis* may be improved by using a medium[2] developed originally for the isolation of spiroplasmas and used subsequently for the isolation of *Mycoplasma genitalium*. Genital mycoplasmas grow well in broth medium under atmospheric conditions, but on agar, colonies develop best in an atmosphere of 95 percent N_2 and 5 percent CO_2. The metabolic activity of mycoplasmas is used to detect their growth in broth medium. Clinical material is added to separate vials of broth-containing phenol red (0.002 percent) and 0.1 percent urea, arginine, or glucose. Ureaplasmas grow best at pH 6.0 or less and possess a urease that breaks down urea to ammonia, thus raising the pH level of the medium so that the color changes from yellow to red. *Mycoplasma hominis* metabolizes arginine to ammonia; thus a similar color change is produced in medium initially at pH 7.0. Glucose-fermenting mycoplasmas cause a decrease in the pH value of the medium that initially is set at 7.5 to 7.8. Aliquots of medium from cultures in which these color changes have occurred are subcultured onto agar medium. Use of this liquid-to-agar technique provides the most sensitive method for the isolation of both ureaplasmas and *M. hominis*.[1] Culturing ureaplasmas takes no more than 1 to 2 days, and M. hominis takes up to about 1 week. However, 1 to 2 months or more may be required to culture *M. genitalium*, the mycoplasma most recently discovered in the genital tract[3] and the respiratory tract.

Ureaplasmas were originally termed *T strains* or *T mycoplasmas* (T for tiny) because they produce very small colonies ranging from 15 to 60 μm in diameter. Colonies of *M. hominis* are about 200 to 300 μm in diameter and have a characteristic "fried egg" appearance (Fig. 1, Chapter 161). Colony size and morphology are, however, not fully reliable as means of identification because increasing the volume of agar and buffering the medium have been shown to increase ureaplasmal colony size, and crowded *M. hominis* colonies may be small and uncharacteristic. On blood agar, *M. hominis*, but not ureaplasmas,

LE 1. Properties of Mycoplasmas Found in the Genital Tract

oplasma	Frequency of Isolation	Metabolism of	Preferred Atmosphere	pH	Hemadsorption	Susceptibility to Thallium	Erythromycin	Lincomycin
realyticum	Common	Urea	Anaerobic	6.0	Serotype 3 only	Yes	Yes	No
ominis	Common	Arginine	~~Aerobic~~ Anaerobic	7.0	No	Yes	No	Yes
ermentans	Rare	Glucose and arginine	Anaerobic	7.5	No	No	Yes	Yes
enitalium	?	Glucose	Anaerobic	7.5	Yes	Yes	Yes	Yes
neumoniae	Very rare	Glucose	Aerobic	7.5	Yes	No	Yes	Yes
rimatum	Rare	Arginine	Anaerobic	7.0	No	No	Yes	Yes
alivarium	Rare	Arginine	Anaerobic	7.0	No	No	Yes	Yes

oduces nonhemolytic pinpoint colonies, and it also grows in ost routine blood culture media without changing their ap- arance. A blind subculture onto blood agar can be used in a agnostic bacteriology laboratory to diagnose bloodstream in- ision with *M. hominis*.[4]

Antibacterial agents, such as penicillin and thallous acetate, e usually added to mycoplasmal media to inhibit bacterial rowth. However, since ureaplasmas, *M. genitalium*, and to a sser extent *M. hominis* are sensitive to thallous acetate,[1,2] it hould be omitted from the media when these organisms are eing sought. Because erythromycin is far more active against reaplasmas than against *M. hominis*, and lincomycin has the everse effect, they may be used to separate the genital my- coplasmas in culture.

Serotyping

There are 14 or more serotypes of *U. urealyticum* and at least seven serotypes of *M. hominis*. Although the data are scanty, none of the work to date has suggested that a particular serotype is convincingly associated with a particular disease.

EPIDEMIOLOGY

Colonization of Infants and Children

Infants usually become colonized with genital mycoplasmas during passage through the infected birth canal; infants delivered by cesarian section are colonized far less often than those delivered vaginally. Ureaplasmas have been isolated from the genitalia of up to one-third of infant girls and *M. hominis* from a smaller proportion.[5,6] The mucosa of the male genital tract is probably less exposed, and this is reflected in the less frequent recovery of mycoplasmas from the genital tract of infant boys.[6] Mycoplasmas, mainly ureaplasmas, have been isolated from the nose and throat of about 15 percent of infants of both sexes.[5] The figures mentioned are estimates and vary from one population to another, depending on the proportion of pregnant women who are colonized.

Neonatal colonization tends not to persist beyond 2 years of age.[6] When mycoplasmas do persist, they do so more often in girls. Thus, genital mycoplasmas have seldom been recovered from prepubertal boys, whereas in one study[7] as many as one-fifth of prepubertal girls were colonized with ureaplasmas and 6 percent with *M. hominis*. In sexually abused children, the organisms are found even more frequently.[8]

Colonization of Adults

After puberty, colonization with genital mycoplasmas occurs primarily as a result of sexual contact.[9,10] This may be deduced from the fact that sexually mature people who have no history of sexual contact are infrequently colonized, whereas colonization among those who are sexually experienced increases in relation to the number of sexual partners. Genital mycoplasmas have been isolated more often from black men and women than from white men and women, but the extent to which these differences are due to differing sexual experience is not clear.

Genital mycoplasmal colonization is also related to socioeconomic status. In Boston, *M. hominis* was isolated from about one-half of the clinic patients at a municipal hospital and ureaplasmas from three-quarters of them, compared to one-fifth and one-half respectively, of the patients visiting private obstetricians and gynecologists in the same area.[11] Whether this apparent socioeconomic difference is a reflection of a difference in sexual experience or whether other factors are involved is unknown. These may include contraception, menstruation, pregnancy, and menopausal changes and have been discussed elsewhere.[12] Genital mycoplasmas appear to be isolated more frequently from pregnant than from nonpregnant women and are isolated less frequently after the menopause, so that the sex hormones may have an influence on colonization.

CLINICAL MANIFESTATIONS

Ureaplasmas and *M. hominis* have been associated with a large variety of clinical conditions,[12,13] as summarized in Table 2, but are considered a cause of only a few conditions.

Nongonococcal Urethritis

There have been numerous studies concerned with the role of mycoplasmas in nongonococcal urethritis (NGU).[12,13] It is clear that most of them (Table 1) cannot be considered as significant causes of NGU because they are isolated so rarely from the genitourinary tract in either healthy or diseased states. *Mycoplasma genitalium* has been detected by a DNA probe in about one-quarter of men with persistent or recurrent NGU and may account for some of these cases.[14] Although *M. hominis* may be isolated from up to 30 percent of patients, the results of numerous studies have failed to implicate it as a cause of the disease.[12,13] On the other hand, several lines of investigation, discussed below, indicate that ureaplasmas are one of the causes of NGU. Their significance has to be evaluated in relation to other microorganisms, particularly *Chlamydia trachomatis* organisms (chlamydiae), which are an undoubted cause.

Isolation Studies. The selection of inappropriate subjects as controls has probably contributed most to the differences among the results of various investigations. NGU patients have been found to harbor ureaplasmas significantly more often than subjects apparently free from disease in about one-half the investigations, whereas the rate of isolation for the NGU and healthy groups has been about the same in the other studies. It is the recovery of ureaplasmas from healthy persons that has been the main source of contention. Most studies have been qualitative, and if ureaplasmas are involved in the pathogenic process, it would be reasonable to expect them to be present in larger numbers than if they were behaving only as commensals. A few workers[15–18] have provided quantitative data to support this idea.

TABLE 2. Association of *Ureaplasma urealyticum* and *Mycoplasma hominis* with Genitourinary and Reproductive Diseases

Disease	*Evidence Suggesting an Association between Indicated Mycoplasma and Disease*		*Evidence Indicating That Mycoplasma Is a Cause of Disease*		Reference Number	Comments on the Relationship and Proportion of Disease Attributable to Mycoplasmas
	U. urealyticum	M. hominis	U. urealyticum	M. hominis		
Nongonococcal urethritis	+ + + +	−	+ + + +	−	3, 12–22, 25, 56	The proportion of NGU caused by ureaplasmas is unknown.
Urethroprostatitis	+ + +	+	+ +	−	3, 12, 13, 16	Ureaplasmas may cause some acute disease, but there is no evidence that they or *M. hominis* cause chronic disease.
Epididymitis	+ + +	−	+ + +	−	12, 13, 46	One case due to ureaplasmas has been described.
Urinary calculi	+ +	−	+ +	−	13, 47	Experimentally, ureaplasmas cause bladder calculi in male rats, and evidence for a cause of natural human disease is increasing.
Pyelonephritis	+	+ + + +	−	+ + + +	13, 26, 27	*M. hominis* causes some cases of acute pyelonephritis and exacerbations.
Reiter's disease	+	−	−	−	12, 13, 22, 36	The significance of ureaplasmas should be assessed further.
Abscess of Bartholin's gland	−	+	−	−	12, 13	Doubtful whether *M. hominis* is involved.
Vaginitis, vaginosis, and cervicitis	−	+ +	−	−	12, 13	*M. hominis* is associated with vaginosis, but a causal relation is unproved.
Pelvic inflammatory disease	+	+ + + +	−	+ + +	12, 13, 28–31	*M. hominis* probably causes some cases, but the proportion is unknown.
Postabortal fever	−	+ + + +	−	+ + + +	13	*M. hominis* is responsible for some cases, but the proportion is unknown.
Postpartum fever	+ +	+ + + +	+ +	+ + + +	4, 13, 32, 33	*M. hominis* may be a major cause.
Involuntary infertility	+ +	−	+	−	13, 48, 49, 59	Ureaplasmas are associated with reduced sperm motility and with infertility in women associated with a male factor.
Repeated spontaneous abortion and stillbirth	+ +	−	−	−	3, 13, 50, 51	Maternal and fetal infections associated with spontaneous abortion, but a causal relation is unproved.
Chorioamnionitis	+ +	−	+ +	−	13, 52	Increasing evidence in some cases.
Low birth weight	+ +	+	+	−	13, 59	An association exists in some studies, but a causal relation is unproved.

Key: + + + +: strong; + + +: good; + +: moderate; +: weak; −: none.

Antibody Studies. Attempts by most workers to detect antibody responses to ureaplasmal infection have not been very successful.[12] However, responses have been detected in about 50 percent of patients by using a large number of serotypes in the metabolism-inhibition test or by employing the enzyme-linked immunosorbent assay (ELISA).[19]

Antibiotic Studies. Some antibiotic studies have been helpful in assessing the role of ureaplasmas in NGU.[12,13] For example, (*1*) in a placebo-controlled trial of minocycline,[20] there was a significant association between minocycline therapy and the resolution of symptoms and signs in patients from whom only ureaplasmas had been isolated. The association was only a little less convincing than that seen between therapy and resolution of disease in patients from whom only chlamydiae had been isolated; in a further study,[21] the best clinical response (96 percent cure rate) to short-term minocycline therapy was seen in men who were experiencing their first attack of NGU and who harbored ureaplasmas only. (*2*) Urethritis in men who harbor chlamydiae and ureaplasmas was unaffected by treatment with differential antibiotics, namely streptomycin and spectinomycin, which eradicate ureaplasmas only; similarly, the disease was unaffected by treatment with sulfafurazole, which eliminates chlamydiae only.[15] Furthermore, more patients responded to minocycline (effective against both microorganisms) than to rifampicin (effective against chlamydiae only), and those infected with ureaplasmas failed to respond to rifampicin significantly more often than those who were not infected.[22] (*3*) About 10 percent of ureaplasmas are resistant to tetracyclines,[17,23,24] and the urethritis of some patients infected by these ureaplasmas is cured only by treatment with antibiotics, such as erythromycin, to which the organisms are susceptible.

Animal and Human Inoculation Studies. Some ureaplasma strains, unpassaged in the laboratory, have produced urethritis and an antibody response in chimpanzees inoculated intra-

urethrally. In addition, three investigators inoculated themselves intraurethrally, and each developed urethritis. Two of them[25] received 5×10^4 ureaplasmas of serotype 5, which had been isolated from patients with NGU who had no other detectable pathogenic microorganisms. The first subject developed urethritis characterized by dysuria, urinary frequency, urethral discomfort, and pyuria. Ureaplasmas were isolated consistently from urine, but they and the associated symptoms and signs disappeared during treatment with minocycline. The second subject also had evidence of mild urethritis and, like the first, a transient antibody response. The predominant feature, however, was the appearance of urinary threads, containing polymorphonuclear leucocytes, which persisted for at least 6 months after treatment with minocycline had eliminated the organisms from meatal, urine, and semen samples.

These various findings suggest that ureaplasmas as well as chlamydiae cause NGU, and, indeed, they would be difficult to explain if ureaplasmas were not involved at all. However, because ureaplasmas may be found in healthy persons, it is important to emphasize that at the present time there is no virtue in subjecting patients with NGU to tests for ureaplasmas on a qualitative routine basis, since positive results, so easy to obtain, are difficult for the clinician to interpret and use in patient management.

Pyelonephritis

Mycoplasma hominis has been isolated, sometimes in pure culture, from the upper urinary tract of almost 10 percent of patients with acute pyelonephritis and antibody to *M. hominis*, measured by the indirect hemagglutination test, has been demonstrated in the serum and urine of some of them. In contrast, recovery has not been achieved from the upper urinary tract of patients with noninfectious urinary diseases, nor has antibody been detected in their urine. Overall, the data[26] suggest that *M. hominis* causes a few cases of acute pyelonephritis or acute exacerbations of chronic pyelonephritis. Ureaplasmas have been recovered very occasionally in the same circumstances and also from aspirates of scarred and renal tissue in patients with reflux nephropathy[27] but their role is not clear.

Pelvic Inflammatory Disease

Like NGU, nongonococcal pelvic inflammatory disease (PID) does not have a single cause. Numerous investigators[12,13] have considered that infection by genital mycoplasmas might be one cause of PID, and three types of study indicate their involvement.

Isolation Studies. *Mycoplasma hominis* has been prominent among more than a dozen reports of the isolation of large-colony-forming mycoplasmas from inflamed fallopian tubes, tuboovarian abscesses, and pelvic abscesses or fluid. The most revealing studies of PID, however, have been those of Swedish workers[28] who used laparoscopy to confirm the diagnosis and collect specimens. *Mycoplasma hominis* was isolated directly from the fallopian tubes of about 10 percent of women with acute salpingitis, but not from those of women without signs of the disease. Similar observations were made in the United Kingdon in 1986 (CM Stacey, PE Munday, D Taylor-Robinson, unpublished data).

Ureaplasmas have been studied less intensively, but have been isolated directly from the fallopian tubes of only a very small proportion of patients with acute salpingitis, from pelvic fluid, and from a tuboovarian abscess. The significance of these findings is unclear, but it seems that if ureaplasmas have any importance, it is much less than that of *M. hominis*.

Antibody Studies. Complement-fixing antibody to *M. hominis* has been found in greater titers in the sera of some patients with salpingitis than in those of others serving as controls. Swedish workers[29] used the more sensitive indirect hemagglutination technique and found antibody to *M. hominis* in about one-half of their patients with salpingitis but in only 10 percent of healthy women. Furthermore, a significant rise or fall in antibody titer occurred during the course of the disease in more than one-half of the women who had *M. hominis* in the lower genital tract. Other workers[30] found that patients with gonococcal PID were more likely to respond serologically to *M. hominis* than those without such disease: they suggested that damage caused by the other organisms was a factor in the response and questioned the primary role of *M. hominis*.

Antibody responses to ureaplasmas in patients with PID have been detected less often than responses to *M. hominis*. This is consistent with the impression that ureaplasmas are less important than *M. hominis* in this disease, although the greater difficulty of detecting ureaplasmal antibody responses must not be forgotten.

Organ Culture and Animal Inoculation Studies. The effect of microorganisms on cells may be examined in organ cultures in which tissues can be maintained almost as in vivo. In fallopian tube organ culturres, *Neisseria gonorrhoeae* rapidly destroys the epithelium, causing complete loss of ciliary activity, whereas *M. hominis*, although multiplying, usually produces little more than swelling of some of the cilia. No damage by ureaplasmas of human origin has been detected.[12,13] This decreasing grade of effect may be a true reflection of the pathogenicity of these microorganisms in vivo, but lack of damage in organ culture does not mean necessarily that the organisms are avirulent. The host immune systems, absent in organ culture, may contribute to pathogenesis, and studies in intact animals may be helpful. It is of interest, therefore, that the introduction of *M. hominis* into grivet monkey oviducts has resulted in a self-limiting acute salpingitis and parametritis with an antibody response,[31] whereas ureaplasmas have had no effect.

The various data strongly suggest that *M. hominis* has a role in causing some cases of acute PID, although the extent to which it behaves as a primary pathogen is still not clear.

Postabortal and Postpartum Fever

Mycoplasma hominis has been isolated from the blood of about 10 percent of women who have fever after abortion, but not from afebrile women who have abortions or from normal pregnant women. In addition, a rise in the titer of *M. hominis* antibody has been detected in about one-half of the women who become febrile, but in only a small proportion of those who have abortions and remain afebrile. Thus, the evidence[13] indicates that *M. hominis* causes some cases of postabortal fever. There is, however, no evidence to suggest that ureaplasmas do likewise. The patients usually recover rapidly without appropriate antimycoplasmal treatment.

After normal vaginal delivery, genital mycoplasmas may be found almost immediately and transiently in the blood of less than 10 percent of women, unassociated with postpartum fever. However, there have been many reports[13] of individual patients with postpartum fever from whose blood *M. hominis* has been isolated a day or more after delivery and in whom an antibody response has been detected.[4] It seems that the organisms may be isolated from the blood of about 5 to 10 percent of such women. Since genital mycoplasmas seldom are recovered from the blood of afebrile women one or more days after delivery, it appears that *M. hominis* induces postpartum fever, presumably by causing endometritis.[32] As in postabortal fever, the patients have a low-grade fever for a day or two after delivery, are not severely ill, and usually recover uneventfully without antibiotic therapy.[4] The role of ureaplasmas is less clear, but they may also be involved.[33]

Hypogammaglobulinemia and Immunosuppression

Some hypogammaglobulinemic patients have developed a chronic urethrocystitis that seemed to be caused by a persistent ureaplasmal and/or *M. hominis* infection, other microorganisms not being isolated.[34] In one case of chronic NGU, the very large number of ureaplasmas recovered persistently from the urethra suggested a causal relationship.[35] Easier to establish is the mycoplasmal etiology of arthritis seen in some patients with hypogammaglobulinemia.[36] This possibility should be considered in any such patient who develops an abacterial septic arthritis. Ureaplasmas and *M. hominis* have been isolated from synovial fluids of a small proportion of these patients, and *M. hominis* has been isolated very occasionally after childbirth in otherwise normal mothers who develop arthritis of sudden onset.[36] The arthritis responds to tetracyclines or other antibiotics to which the organisms are sensitive, a further indication that they are a cause of the disease.

Immunosuppression may lead to proliferation of mycoplasmas, and septicemia,[37] and peritonitis[38] due to *M. hominis* have been recorded.

Other Diseases

The few reported cases of neonatal meningitis or brain abscess in which *M. hominis* has been isolated from cerebrospinal fluid[39–41] or abscess[42,43] have resulted presumably from infection in utero or from colonization at birth with subsequent infection. The same comment applies to the recovery of ureaplasmas from cerebrospinal fluid.[41] The possibility should be considered in cases of neonatal central nervous system disease in which the results of bacteriologic staining and culture are negative. Apart from fever following abortion or normal childbirth, fever associated with burns and trauma has also been attributed to *M. hominis* infection,[37,44] and this mycoplasma has also been implicated in some wound infections.[45]

Conditions of Rare or Equivocal Mycoplasmal Etiology

As shown in Table 2, there are various conditions, such as epididymitis, urinary calculi, Reiter disease, infertility, spontaneous abortion, chorioamnionitis, and low birth weight, with which ureaplasmas, in particular, have been associated. In some instances the association is rare, and in others there is insufficient proof or no proof that the organisms are a cause. Recent observations indicate that ureaplasmas are a rare cause of acute nonchlamydial epididymitis, the organisms having been recovered from the epididymis in association with an antibody response.[46] Furthermore, the results of studies on ureaplasmas in urinary calculi,[47] infertility,[48] particularly among a subgroup of infertile women whose problem is associated with a male factor,[49] abortion,[50,51] chorioamnionitis,[52] and respiratory disease in the newborn,[53] including the respiratory distress syndrome,[54] and chronic lung disease[55] are provocative and should stimulate further work to define their role.

MANAGEMENT AND TREATMENT

Since culturing for mycoplasmas is not generally available to clinicians, management depends on recognizing clinical syndromes for which mycoplasmas could be responsible and providing therapy that would be adequate to eliminate them.

The weight of accumulated evidence suggests that both *C. trachomatis* and *U. urealyticum* cause NGU. Patients should receive a tetracycline, for example doxycycline, 100 mg twice daily for 7 days. However, about 10 percent of ureaplasmas are resistant to tetracyclines,[17,23,24] and patients with NGU due to resistant organisms often have no clinical response to the administration of a tetracycline.[56] In this circumstance, the patients should be examined for tetracycline-resistant ureaplasmas if laboratory facilities are available. Meanwhile, they should be treated with erythromycin, 0.5 g four times daily for 7 days, since most tetracycline-resistant ureaplasmas are sensitive to this antibiotic.

It would be advisable to treat PID with a tetracycline in areas where a substantial proportion of the disease is nongonococcal, since tetracyclines are active against most strains of *M. hominis* as well as against *C. trachomatis*, which also causes PID. However, the emergence of tetracycline-resistant strains of *M. hominis*[36,40,57] means that other antibiotics, such as lincomycin or clindamycin, may need to be considered. If *M. hominis*-induced fever following abortion or vaginal delivery does not settle rapidly, tetracycline therapy should be instituted while keeping tetracycline resistance in mind. The latter assumes greater importance in other clinical situation, such as arthritis and neonatal disease, where *M. hominis* is considered to be responsible.

Antibiotic treatment for infertility,[58] spontaneous abortion,[50] or low birth weight[59] is obviously legitimate on a research basis. However, for these and other conditions in which a mycoplasmal etiology has not been proved, it is difficult to justify either examination for the organisms or treatment directed against them on a routine basis. Culture of genital specimens from adults with an idiopathic disorder results in the isolation of either ureaplasmas or *M. hominis*, or both, from about one-half of them. To consider the organisms a cause of the disorder on the basis of such a predictable microbiologic finding is not warranted, and to provide routine antibiotic therapy aimed at the mycoplasmas in such instances would seem as unethical as not initiating effective treatment when the etiology is understood.

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SECTION D. RICKETTSIOSIS

164. INTRODUCTION

ALFRED J. SAAH

The family of microbes Rickettsiaceae is maintained in nature through a cycle involving reservoirs in mammals and insect vectors. The public health impact on lives or productivity lost is largely unmeasured, but it is suspected to be quite high worldwide.[1] Humans are incidental hosts and are not useful in propagating the organism in nature. An exception is louse-borne typhus where humans are the principal reservoir and the human body louse is the vector, thereby creating a cycle that involves humans alone. However, even louse-borne typhus may also prove to be a zoonotic disease. Data have been reported implicating the flying squirrel as a reservoir of the agent that produces louse-borne typhus,[2,3] and serologic evidence in humans suggests that louse-borne typhus occurs indigenously in the United States[4,5] (see Chapter 168).

DESCRIPTION OF THE PATHOGEN

Rickettsiae are fastidious bacterial organisms that are obligate, intracellular parasites. The organisms are small, pleomorphic coccobacilli. Coccal forms usually are 0.3 μm in diameter, while bacillary forms measure 0.3 μm × 1.0–2.0 μm. The bacterial

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